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LACTOGENS	76
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(L10 AND LACTOGEN).USPT,PGPB,JPAB,EPAB,DWPI,TDBD.	28

Database:

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Derwent World Patents Index
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L11

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DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=YES; OP=ADJ

<u>L11</u>	L10 and lactogen	28	<u>L11</u>
<u>L10</u>	L9 and interferon	65	<u>L10</u>
<u>L9</u>	hpl	1009	<u>L9</u>
<u>L8</u>	isplp	2	<u>L8</u>
<u>L7</u>	peyman-john-a.in.	16	<u>L7</u>
<u>L6</u>	interferon and supress\$3 and placent\$3 and lactogen	15	<u>L6</u>
<u>L5</u>	interferon and supress\$3 placent\$3	0	<u>L5</u>
<u>L4</u>	interferon and supress\$3	571	<u>L4</u>
<u>L3</u>	interferon and supress\$3 placent\$3 and lactogen	0	<u>L3</u>
<u>L2</u>	interferon and supress\$3 placent\$3 and lactogen and peptide	0	<u>L2</u>
<u>L1</u>	interferon-supressing placental lactogen peptide	0	<u>L1</u>

END OF SEARCH HISTORY

*****STN Columbus*****

FILE 'MEDLINE'
FILE 'JAPIO'
FILE 'BIOSIS'
FILE 'SCISEARCH'
FILE 'WPIDS'

=> s interferon and suppressing and placental and lactogen and peptide
L1 0 INTERFERON AND SUPPRESSING AND PLACENTAL
AND LACTOGEN AND PEPTIDE

=> s interferon-suppressing
L2 0 INTERFERON-SUPPRESSING

=> s interferon suppressing
L3 0 INTERFERON SUPPRESSING

=> s interferon suppressing
1 FILES SEARCHED...
L4 9 INTERFERON SUPPRESSING

=> s interferon and suppressing and placental and lactogen and peptide
L5 2 INTERFERON AND SUPPRESSING AND PLACENTAL
AND LACTOGEN AND PEPTID
E

=> isplp
L6 2 ISPLP

=> s isplp
L7 2 ISPLP

=> l7 and interferon
L8 2 L7 AND INTERFERON

=> s interferon and inhibiting
L9 8203 INTERFERON AND INHIBITING

=> l9 and lactogen
L10 11 L9 AND LACTOGEN

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11 9 DUP REM L10 (2 DUPLICATES REMOVED)

=> dup rem l8
PROCESSING COMPLETED FOR L8
L12 1 DUP REM L8 (1 DUPLICATE REMOVED)

=> dup rem l7
PROCESSING COMPLETED FOR L7
L13 1 DUP REM L7 (1 DUPLICATE REMOVED)

=> dup rem l5
PROCESSING COMPLETED FOR L5
L14 1 DUP REM L5 (1 DUPLICATE REMOVED)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L15 4 DUP REM L4 (5 DUPLICATES REMOVED)

=> d ibib abs l15 1-4

L15 ANSWER 1 OF 4 WPIDS COPYRIGHT 2003 THOMSON
DERWENT ON STN DUPLICATE 1

ACCESSION NUMBER: 2002-424996 [45] WPIDS
DOC. NO. CPI: C2002-120339
TITLE: ***interferon*** - ***suppressing*** placental

lactogen peptides, useful for treating autoimmune
disease, inflammatory disease or organ transplant
rejection, e.g. lupus erythematosus, Crohn's disease,
eczema, septic shock or ischemia.

DERWENT CLASS: B04
INVENTOR(S): PEYMAN, J A
PATENT ASSIGNEE(S): (PEYM-I) PEYMAN J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO KIND DATE WEEK LA PG
.....
US 2002032154 A1 20020314 (200245)* 31

APPLICATION DETAILS:

PATENT NO KIND APPLICATION DATE
.....
US 2002032154 A1 Provisional US 2000-210082P 20000607
US 2001-876478 20010607

PRIORITY APPLN. INFO: US 2000-210082P 20000607; US
2001-876478

20010607

AN 2002-424996 [45] WPIDS

AB US2002032154 A UPAB: 20020717

NOVELTY - ***interferon*** - ***suppressing*** placental
lactogen

peptides which suppress IFN-gamma-stimulated expression of MHC
class II

antigens, MHC class I antigens and/or ICAM-1 antigen, are new.

DETAILED DESCRIPTION - An ***interferon*** -

suppressing

placental lactogen peptide (which suppresses IFN-gamma-stimulated
expression of MHC class II antigens MHC class I antigens and/or

antigen) comprises:

(a) N-terminal 28 residues of hPL, or hPL-1;
(b) a 28-amino acid sequence with substantial identity to hPL or
hPL-1, containing one or more conservative amino acid substitutions;
(c) a derivative of hPL or hPL-1, having residues 5-27; or
(d) a derivative of (b) having residues 5-27.

The ***interferon*** - ***suppressing*** placental lactogen
peptides (ISPLP) hPL and hPL-1 have the sequences S1 and S2
respectively.

INDEPENDENT CLAIMS are also included for:

(1) treating a human subject by administering to the subject a cell
or tissue that has been treated with the peptide; and
(2) treating a human subject by administering the ISPLP.

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Ala-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Iys-Glu-Ala-Met-Leu-Gln-Ala-
His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)

ACTIVITY - Immunosuppressive; Antiinflammatory;

Neuroprotective;

Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.

No biological data given.

MECHANISM OF ACTION - Interferon gamma suppressor.

USE - The peptide is useful for treating autoimmune disease,
inflammatory disease or organ transplant rejection (claimed). These
diseases include lupus erythematosus, multiple sclerosis, Crohn's
disease,

asthma, hay fever, eczema, sepsis, septic shock or ischemia.

Dwg.9/11

L15 ANSWER 2 OF 4 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 96207396 MEDLINE

DOCUMENT NUMBER: 96207396 PubMed ID: 8615043

TITLE: NF-kappaB activation is delayed in mouse L929 cells
infected with ***interferon*** ***suppressing*** ,
but not inducing, vesicular stomatitis virus strains.

AUTHOR: Boulares A H; Ferran M C; Lucas-Lenard J

CORPORATE SOURCE: Molecular and Cell Biology Department,
University of

Connecticut, Storrs, 06269-3125, USA.

SOURCE: VIROLOGY, (1996 Apr 1) 218 (1) 71-80.

Journal code: 0110674. ISSN: 0042-6822.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199606

ENTRY DATE: Entered STN: 19960613

Last Updated on STN: 19980206

Entered Medline: 19960605

AB Vesicular stomatitis virus (VSV) mutant T1026R1 of the Indiana (IN)
serotype is a good inducer of interferon (IFN). This mutant was used to
study the activation of NF-kappaB, a transcription factor necessary for
IFN induction, in mouse L929 cells that were stably transfected with a
chimeric gene containing the human IFN-beta gene promoter attached
to the
chloramphenicol acetyltransferase (CAT) coding sequence. NF-kappaB
DNA

binding activity was detected as early as 30 min after virus adsorption in
nuclear extracts, increased up to 4 hr, and then remained constant for at
least 6 additional hr. The kinetics of CAT expression correlated with the
kinetics of NF-kappaB nuclear DNA binding activity. Virus entry and
delivery of viral components into the cytoplasm were required for
NF-kappaB activation. Exposure of T1026R1 to one hit of UV

irradiation
nearly completely reduced NF-kappaB activation. In cells infected with
wild-type (wt) VSV (IN), a noninducer of IFN, NF-kappaB DNA
binding
activity in the nucleus was delayed for several hours after virus
adsorption. Coinfection of wt VSV and T1026R1 resulted in the
reduction
of T1026R1-promoted NF-kappaB activation. This inhibitory activity
of wt
VSV was abolished by one hit of UV irradiation. Under similar
conditions
expression of the CAT gene was more UV resistant, suggesting that
IFN gene
expression is regulated at multiple levels.

L15 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL
ABSTRACTS INC. on STN

ACCESSION NUMBER: 1990-4193 BIOSIS

DOCUMENT NUMBER: PREV199089004193; BA89:4193

TITLE: INTERFERON INDUCTION BY VIRUSES XVIII.
VESICULAR STOMATITIS

VIRUS NEW JERSEY A SINGLE INFECTIOUS

PARTICLE CAN BOTH

INDUCE AND SUPPRESS INTERFERON PRODUCTION.

AUTHOR(S): GACCIONE C (Reprint author); MARCUS P I

CORPORATE SOURCE: DEP MOI. CELL BIOL, UNIV

CONNECTICUT, STORRS, CONN

06269-3044, USA

SOURCE: Journal of Interferon Research, (1989) Vol. 9, No. 5,
pp.

603-614.

CODEN: JIREJD. ISSN: 0197-8357.

DOCUMENT TYPE: Article

FILE SEGMENT: BA

LANGUAGE: ENGLISH

ENTRY DATE: Entered STN: 5 Dec 1989

Last Updated on STN: 5 Dec 1989

AB In contrast to wild-type vesicular stomatitis virus (VSV) of Indiana
(Ind) origin which express interferon (IFN) inducing- and IFN

individuals particles of wild-type VSV of the New Jersey (N.J.)

serotype

(Hazelhurst [H] isolate) paradoxically can both induce IFN and
suppress

its induction in cells coinfectd with a potent inducer of IFN. The
properties of IFN induction, and its suppression, appear to reside in the
particle that manifests infectivity. Analyses of IFN induction
dose-response curves to measure IFN-inducing particles (IFP), and IFN
yield-reduction curves to measure IFN induction-suppressing particles
(ISP) generated by VSV-N.J.(H) in aged chick embryo cells revealed

that

(i) a single particle per cell sufficed to induce a quantum (full) yield
of IFN, or to suppress fully IFN production by a coinfecting inducing
virus, and (ii) the addition of one or more IFP per cell did not suppress
the yield of IFN beyond the plateau level. The time-course of IFN
production in chick cells infected with VSV-N.J.(H) revealed about a
4-h

lag, even when the cells were coinfectd with a potent inducer that
normally induced IFN 1 or 2 h sooner. Thus, VSV-N.J.(H) appears to
regulate the production of IFN in cells-even that initiated by other
inducers. Expression of IFP and ISP activities both required primary
transcription, with respective genomic targets similar to those reported
for VSV-Ind. N.J.(H) is the first wild-type VSV observed to express
IFP

and ISP activities concomitantly. A model is presented to suggest how
these two antagonistic properties might be expressed by a single
infectious particle.

L15 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL
ABSTRACTS INC. on STN

ACCESSION NUMBER: 1989-353051 BIOSIS

DOCUMENT NUMBER: PREV198988045165; BA88:45165

TITLE: INFLUENCE OF INTERFERON CIRCULATING IN

THE BLOOD OF INTACT

GUINEA-PIGS ON THE FORMATION OF AN

IMMEDIATE ALLERGY.

AUTHOR(S): ERMEKOVA D M (Reprint author); ASPETOV R D;

ABDRASILOVA G S

CORPORATE SOURCE: RES INST EPIDEMIOI. MICROBIOL

INFECT DIS, MINIST HEALTH KAZ.

SSR, ALMA-ATA, USSR

SOURCE: Immunologiya, (1988) No. 6, pp. 57-58.

ISSN: 0206-4952.

DOCUMENT TYPE: Article

FILE SEGMENT: BA

LANGUAGE: RUSSIAN

ENTRY DATE: Entered STN: 2 Aug 1989

Last Updated on STN: 2 Aug 1989

AB The data are presented on interferon detection in the blood serum of
intact guinea pigs and influence of gamma -interferon on formation fo
immediate allergy to Artemisia scoparia pollen. It has been found that
the circulating blood of a great number of guinea pigs contains
gamma -

interferon ***suppressing*** the process of formation of
immediate response to Artemisia scoparia pollen allergens.

=> d ibib abs l14

L14 ANSWER 1 OF 1 WPIDS COPYRIGHT 2003 THOMSON
DERWENT ON STN DUPLICATE 1

ACCESSION NUMBER: 2002-424996 [45] WPIDS

DOC. NO. CPI: C2002-120339

TITLE: ***interferon*** - ***suppressing***
placental ***lactogen*** ***peptides*** ,
useful for treating autoimmune disease, inflammatory
disease or organ transplant rejection, e.g. lupus
erythematosus, Crohn's disease, eczema, septic shock or
ischemia.

DERWENT CLASS: B04
INVENTOR(S): PEYMAN, J A
PATENT ASSIGNEE(S): (PEYM-I) PEYMAN J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO KIND DATE WEEK LA PG
.....
US 2002032154 A1 20020314 (200245)* 31

APPLICATION DETAILS:

PATENT NO KIND APPLICATION DATE
.....
US 2002032154 A1 Provisional US 2000-210082P 20000607
US 2001-876478 20010607

PRIORITY APPLN. INFO: US 2000-210082P 20000607; US
2001-876478

20010607

AN 2002-424996 [45] WPIDS

AB US2002032154 A UPAB: 20020717

NOVELTY - ***interferon*** - ***suppressing***

placental

lactogen ***peptides*** which suppress

IFN-gamma-stimulated
expression of MHC class II antigens, MHC class I antigens and/or
ICAM-1

antigen, are new.

DETAILED DESCRIPTION - An ***interferon*** -

suppressing

placental ***lactogen*** ***peptide*** (which

suppresses

IFN-gamma-stimulated expression of MHC class II antigens, MHC
class I

antigens and/or ICAM-1 antigen) comprises:

(a) N-terminal 28 residues of hPL, or hPL-1;

hPL-1, containing one or more conservative amino acid substitutions;
(c) a derivative of hPL or hPL-1, having residues 5-27; or
(d) a derivative of (b) having residues 5-27.
The ***interferon*** - ***suppressing*** ***placental***
lactogen ***peptides*** (ISPLP) hPL and hPL-1 have the
sequences S1 and S2 respectively.
INDEPENDENT CLAIMS are also included for:
(1) treating a human subject by administering to the subject a cell
or tissue that has been treated with the ***peptide***; and
(2) treating a human subject by administering the ISPLP.

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Al
a- His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Lys-Glu-Ala-Met-Leu-Gln-Al
a- His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)
ACTIVITY - Immunosuppressive; Antiinflammatory;
Neuroprotective;
Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.
No biological data given.
MECHANISM OF ACTION - ***Interferon*** gamma
suppressor.

USE - The ***peptide*** is useful for treating autoimmune
disease, inflammatory disease or organ transplant rejection (claimed).
These diseases include lupus erythematosus, multiple sclerosis, Crohn's
disease, asthma, hay fever, eczema, sepsis, septic shock or ischemia.
Dwg.0/11

> d ibib abs 113

L13 ANSWER 1 OF 1 WPIDS COPYRIGHT 2003 THOMSON

DERWENT on STN DUPLICATE 1
ACCESSION NUMBER: 2002-424996 [45] WPIDS
DOC. NO. CPI: C2002-120339
TITLE: Interferon-suppressing placental lactogen peptides,
useful for treating autoimmune disease, inflammatory
disease or organ transplant rejection, e.g. lupus
erythematosus, Crohn's disease, eczema, septic shock or
ischemia.

DERWENT CLASS: B04
INVENTOR(S): PEYMAN, J A
PATENT ASSIGNEE(S): (PEYM-I) PEYMAN J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002032154 A1	20020314	(200245)*	31		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002032154 A1	Provisional	US 2000-210082P	20000607
		US 2001-876478	20010607

PRIORITY APPLN. INFO: US 2000-210082P 20000607; US
2001-876478

AN 2002-424996 [45] WPIDS
AB US2002032154 A UPAB: 20020717
NOVELTY - Interferon-suppressing placental lactogen peptides which
suppress IFN-gamma-stimulated expression of MHC class II antigens,
MHC

class I antigens and/or ICAM-1 antigen, are new.
DETAILED DESCRIPTION - An interferon-suppressing placental
lactogen

peptide (which suppresses IFN-gamma-stimulated expression of MHC
class II
antigens, MHC class I antigens and/or ICAM-1 antigen) comprises:
(a) N-terminal 28 residues of hPL, or hPL-1;
(b) a 28-amino acid sequence with substantial identity to hPL or
hPL-1, containing one or more conservative amino acid substitutions;
(c) a derivative of hPL or hPL-1, having residues 5-27; or
(d) a derivative of (b) having residues 5-27.

The interferon-suppressing placental lactogen peptides (***ISPLP***
) hPL and hPL-1 have the sequences S1 and S2 respectively.
INDEPENDENT CLAIMS are also included for:
(1) treating a human subject by administering to the subject a cell
or tissue that has been treated with the peptide; and
(2) treating a human subject by administering the ***ISPLP***.

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Al
a- His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Lys-Glu-Ala-Met-Leu-Gln-Al
a- His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)
ACTIVITY - Immunosuppressive; Antiinflammatory;
Neuroprotective;
Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.
No biological data given.
MECHANISM OF ACTION - Interferon gamma suppressor.
USE - The peptide is useful for treating autoimmune disease,
inflammatory disease or organ transplant rejection (claimed). These
diseases include lupus erythematosus, multiple sclerosis, Crohn's
disease,
asthma, hay fever, eczema, sepsis, septic shock or ischemia.
Dwg.0/11

L12 ANSWER 1 OF 1 WPIDS COPYRIGHT 2003 THOMSON
DERWENT on STN DUPLICATE 1
ACCESSION NUMBER: 2002-424996 [45] WPIDS
DOC. NO. CPI: C2002-120339
TITLE: ***Interferon*** -suppressing placental lactogen
peptides, useful for treating autoimmune disease,
inflammatory disease or organ transplant rejection, e.g.
lupus erythematosus, Crohn's disease, eczema, septic
shock or ischemia.

DERWENT CLASS: B04
INVENTOR(S): PEYMAN, J A
PATENT ASSIGNEE(S): (PEYM-I) PEYMAN J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002032154 A1	20020314	(200245)*	31		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002032154 A1	Provisional	US 2000-210082P	20000607
		US 2001-876478	20010607

PRIORITY APPLN. INFO: US 2000-210082P 20000607; US
2001-876478

AN 2002-424996 [45] WPIDS
AB US2002032154 A UPAB: 20020717
NOVELTY - ***Interferon*** -suppressing placental lactogen
peptides
which suppress IFN-gamma-stimulated expression of MHC class II
antigens,
MHC class I antigens and/or ICAM-1 antigen, are new.
DETAILED DESCRIPTION - An ***interferon*** -suppressing
placental
lactogen peptide (which suppresses IFN-gamma-stimulated expression
of MHC
class II antigens, MHC class I antigens and/or ICAM-1 antigen)
comprises:
(a) N-terminal 28 residues of hPL, or hPL-1;
(b) a 28-amino acid sequence with substantial identity to hPL or
hPL-1, containing one or more conservative amino acid substitutions;
(c) a derivative of hPL or hPL-1, having residues 5-27; or
(d) a derivative of (b) having residues 5-27.
The ***interferon*** -suppressing placental lactogen peptides (***ISPLP***) hPL and hPL-1 have the sequences S1 and S2
respectively.

INDEPENDENT CLAIMS are also included for:
(1) treating a human subject by administering to the subject a cell
or tissue that has been treated with the peptide; and
(2) treating a human subject by administering the ***ISPLP***.

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Asp-His-Ala-Met-Leu-Gln-Al
a- His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S1)

Val-Gln-Thr-Val-Pro-Leu-Ser-Arg-Leu-Phe-Lys-Glu-Ala-Met-Leu-Gln-Al
a- His-Arg-Ala-His-Gln-Leu-Ala-Ile-Asp-Thr-Tyr (S2)
ACTIVITY - Immunosuppressive; Antiinflammatory;
Neuroprotective;
Antiasthmatic; Antiallergic; Dermatological; Antibacterial; Vasotropic.
No biological data given.
MECHANISM OF ACTION - ***Interferon*** gamma
suppressor.

USE - The peptide is useful for treating autoimmune disease,
inflammatory disease or organ transplant rejection (claimed). These
diseases include lupus erythematosus, multiple sclerosis, Crohn's
disease,
asthma, hay fever, eczema, sepsis, septic shock or ischemia.
Dwg.0/11

=> d ibib abs 111 1-9

L11 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003-796985 CAPLUS
DOCUMENT NUMBER: 139:287284

TITLE: Solution phase hybridization-based methods for
detecting and quantitating nucleic acid analytes
INVENTOR(S): Stephan, Jean-Philippe F.; Tsai, Siao Ping; Wong,
Wai

Lee Tan; Billeci, Todd
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 94 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003083440	A2	20031009	WO 2003-US9726	20030328
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ,				
CA, CH, CN,				
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD,				
GE, GH,				
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,				
LR,				
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI,				
NO, NZ, OM				

TT,
TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY,
KG, KZ, MD,
RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW,
AT, BE, BG,
CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU,
MC,
NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
GQ,
GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO: US 2002-368669P P 20020329
AB The present invention provides novel soln. phase hybridization-based
methods for detecting and quantitating nucleic acid analytes. Methods
comprising use of novel capture polymers and/or signaling systems are
provided. Use of these novel capture polymers and/or signaling systems
provides significant improvements in signal to noise ratio, specificity,
sensitivity and ease of development and use as compared to existing
soln.
phase nucleic acid detection and quantitation methods. The invention
further provides compns., kits and articles of manuf. for practicing
methods of the present invention.

L11 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:390758 CAPLUS
DOCUMENT NUMBER: 138:390934

TITLE: Topical and transdermal administration of peptidyl
drugs with hydroxide-releasing agents as skin
permeation enhancers
INVENTOR(S): Luo, Eric C.; Jacobson, Eric C.; Hsu, Tsung-Min
PATENT ASSIGNEE(S): Dermatrends, Inc., USA
SOURCE: U.S., 13 pp., Cont.-in-part of U.S. Ser. No. 569,889.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 23
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6565879	B1	20030520	US 2000-687937	20001013
US 2001038862	A1	20011108	US 2000-737831	20001214
US 6558695	B2	20030506		
US 2003147943	A1	20030807	US 2003-349582	20030122
PRIORITY APPLN. INFO:			US 1999-465098	A2 19991216
			US 2000-569889	A2 20000511
			US 2000-687937	A2 20001013
			US 2000-737831	A3 20001214

AB A method is provided for increasing the permeability of skin or
mucosa)
tissue to a topically or transdermally administered pharmacol. or
cosmeceutically active peptide, polypeptide or protein. The method
involves use of a specified amt. of a hydroxide-releasing agent, the amt.
optimized to increase the flux of the peptide, polypeptide or protein
through a body surface while minimizing the likelihood of skin damage.
irritation or sensitization. Formulations and drug delivery devices
employing hydroxide-releasing agents as permeation enhancers are
provided
as well. An in vitro human cadaver skin permeation study was
conducted
using 0.18% leuprolide soln. and 3.6% sodium hydroxide.
REFERENCE COUNT: 39 THERE ARE 39 CITED
REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE
RE FORMAT

L11 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:332611 CAPLUS
DOCUMENT NUMBER: 136:337369

TITLE: Real time quantitative aptamer-PCR or immuno-PCR
with
detectable non-primer probes
INVENTOR(S): Dodge, Anthony H.; Meng, Yu-Ju G.; Sims, Paul
W.;

Sinicropi, Dominick V.; Williams, P. Mickey; Wong, Wai
Lee
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 37 pp.

CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002051974	A1	20020502	US 1999-449204	19991124
PRIORITY APPLN. INFO:			US 1998-110259P	P 19981130
AB The invention relates to a method for a novel method for detecting the presence of a target compd., in a sample which may contain the target compd., using a nucleic acid detector mol., amplification and quantitation or detection of the detector mol. The method uses the following steps: (a) exposing a sample, which may contain or is suspected of contg. the target compd., to a capture mol. capable of binding to the target mol. to form a capture mol.: target mol. complex; (b) adding to the capture mol.: target mol. complex, a detector mol. contg. a nucleic acid moiety and capable of specifically binding to the target mol.; and (c) amplifying the nucleic acid moiety by PCR amplification, and (d) quantitating or detecting the PCR amplified nucleic acid moiety using a detectable non-primer probe capable of binding to the nucleic acid moiety. In the method of the invention, the capture mol. may be an antibody, phage antibody, aptamer or other receptor or binding partner for the analyte of interest. The detector mol. may be either a nucleic acid labeled antibody				

achieved by detecting the amplified nucleic acid (nucleic acid moiety on the labeled antibody or aptamer) with a detectable non-primer probe capable of binding to the amplified nucleic acid, preferably in real time. The invention, therefore, provides improvements in quantitation and sensitivity over immuno-PCR and ELONA assays which have been used

protein analytes, by utilizing a PCR amplification and quantification technique used only for application to the real time detection of nucleic acids. The invention also provides improvements over conventional ELISA

assays in sensitivity. The use of non-primer probes, preferably with real time anal., e.g., the TaqMang system, in an aptamer-PCR or an immuno-PCR

assay as in the invention, overcomes the shortcomings of prior art processes discuss above. Since the PCR reaction products are not subjected to post-PCR manipulations, the risk of product contamination

in assays is significantly lowered. Monitoring the PCR reaction in 'real time' allows the collection of data across many cycles (e.g. cycle 1-50) instead of at an endpoint PCR stage, as in conventional immuno-PCR (e.g.

cycle 25 only), therefore allowing for a greater range of detectable amplicon. The method of the invention can detect the target mol. at a concn. of less than 1.0x10⁻¹-g/mL, generally about 1.0x10⁻¹⁵ to about 1.0x10⁻⁸ g/mL. Detecting vascular endothelial growth factor (VEGF)

using the aptamer rt-PCR assay of the invention is described.

L11 ANSWER 4 OF 9 WPIDS COPYRIGHT 2003 THOMSON DERWENT ON STN DUPLICATE 1

ACCESSION NUMBER: 2002-082332 [11] WPIDS CROSS REFERENCE: 2001-441621 [47]; 2001-488538 [53]; 2002-065878 [09];

2002-074630 [10]; 2002-082296 [11]; 2002-105506 [14]; 2002-470580 [50]; 2003-341108 [32]; 2003-361827 [34]; 2003-370772 [35]; 2003-370773 [35]; 2003-456180 [43]; 2003-531377 [50]; 2003-554600 [52]; 2003-644797 [61]; 2003-658676 [62]; 2003-669970 [63]; 2003-670893 [63]; 2003-677996 [64]; 2003-720863 [68]; 2003-720864 [68]

DOC. NO. CFI: C2002-024825

TITLE: Composition useful for delivering peptidyl drugs comprises a hydroxide releasing agent.

DERWENT CLASS: B04 D16 P32 P34

INVENTOR(S): HSU, T, LUO, E C

PATENT ASSIGNEE(S): (HSU-T) HSU T; (LUO-E) LUO E C;

(DERM-N) DERMATRENDS

INC

COUNTRY COUNT: 1

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2001038862 A1		20011108 (200211)*	16		
US 6558695 B2		20030506 (200338)			

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2001038862 A1	CIP of	US 1999-465098	19991216
	CIP of	US 2000-569889	20000511
	CIP of	US 2000-687937	20001013
		US 2000-737831	20001214
US 6558695 B2	CIP of	US 1999-465098	19991216
	CIP of	US 2000-569889	20000511
	CIP of	US 2000-687937	20001013
		US 2000-737831	20001214

PRIORITY APPLN. INFO: US 2000-737831 20001214; US 1999-465098

19991216; US 2000-569889 20000511; US 2000-687937 20001013

AN 2002-082332 [11] WPIDS

CR 2001-441621 [47]; 2001-488538 [53]; 2002-065878 [09]; 2002-074630 [10];

2002-082296 [11]; 2002-105506 [14]; 2002-470580 [50]; 2003-341108 [32];

2003-361827 [34]; 2003-370772 [35]; 2003-370773 [35]; 2003-456180 [43];

2003-531377 [50]; 2003-554600 [52]; 2003-644797 [61]; 2003-658676 [62];

2003-669970 [63]; 2003-670893 [63]; 2003-677996 [64]; 2003-720863 [68];

2003-720864 [68]

AB US2001038862 A UPAB: 20031022

NOVELTY - A composition comprises an aqueous formulation of: a peptidyl

drug (I), a hydroxide-releasing agent (II), and a carrier (III). (II) enhances the flux of (I) through the body surface.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a system

for topical or transdermal administration of (I) comprising: at least one reservoir containing (I) and (II); a device for maintaining the system in drug and enhancer transmitting relationship to the body surface; and a backing layer. The backing layer serves as the outer surface of the system

during use.

ACTIVITY - Analgesic.

MECHANISM OF ACTION - ***Interferon*** -therapy.

USE - For the delivery of peptidyl drug through a body surface. The peptidyl drug are useful in variety of diseases e.g. pain.

ADVANTAGE - The composition is substantially free of additional permeation enhancer and organic solvents. The formulation increases the

rate at which an active agent permeates the skin, and does not result in

irritation. (II) are highly effective

toxicity.
Dwg.0/0

L11 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 2001:851194 CAPLUS

DOCUMENT NUMBER: 136:2090

TITLE: Methods for refolding of growth hormone supergene family proteins containing free cysteine residues

INVENTOR(S): Rosendahl, Mary S.; Cox, George N.; Doherty, Daniel H.

PATENT ASSIGNEE(S): Bolder Biotechnology, Inc., USA

SOURCE: PCT Int. Appl., 110 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001087925 A2		20011122	WO 2001-US16088	20010516
WO 2001087925 A3		20020801		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,

HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,

LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,

SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,

YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,

BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG EP 1284987 A2 20030226 EP 2001-941504 20010516

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.: US 2000-204617 P 20000516 WO 2001-US16088 W 20010516

AB The present invention relates to novel methods for making and refolding

insol. or aggregated proteins having free cysteines in which a host cell expressing the protein is exposed to a cysteine blocking agent.

Blocking

of free cysteines prevents the crosslinking of proteins into large insol. aggregates, keeping them in soln. and simplifying purifn. and increasing the yield of the biol. activity. The sol., refolded proteins produced by the novel methods can then be modified to increase their effectiveness.

Such modifications include attaching a PEG moiety to form PEGylated proteins. The PEGylated proteins of the investigation include

recombinant

cysteine variants of members of the growth hormone supergene family such

as: growth hormone, granulocyte colony-stimulating factor, granulocyte macrophage colony-stimulating factor, and alpha- ***interferon***.

L11 ANSWER 6 OF 9 WPIDS COPYRIGHT 2003 THOMSON

DERWENT ON STN DUPLICATE 2

ACCESSION NUMBER: 2000-096723 [08] WPIDS

DOC. NO. CFI: C2000-028026

TITLE: ***Inhibiting*** ceramide-mediated apoptosis of

mammalian cells.

DERWENT CLASS: B04

INVENTOR(S): LEVI, M; SHIMABUKURO, M; UNGER, R H; ZHOU, Y

PATENT ASSIGNEE(S): (TEXA) UNIV TEXAS SYSTEM

COUNTRY COUNT: 84

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9944598 A2		19990910 (200008)* EN 77			
RW: AT BE CH CY DE DK EA ES FI FR GB GH GJ GM GR IE IT					

KE LS LU MC MW NL

OA PT SD SE SL SZ UG ZW

W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GR

GE GH GM HD HU ID IL IN IS JP KE KG KP KR KZ LC LK

LR LS LT LU LV

MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI

SK SL TJ TM TR TT

UA UG US UZ VN YU ZW

AU 9928931 A 19990920 (200008)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9944598 A2		WO 1999-US4730	19990303
AU 9928931 A		AU 1999-28931	19990303

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 9928931 A	Based on	WO 9944598

PRIORITY APPLN. INFO: US 1998-76818P 19980303

AN 2000-096723 [08] WPIDS

AB WO 9944598 A UPAB: 20000215

NOVELTY - A method of ***inhibiting*** ceramide-mediated apoptosis in

reduces ceramide levels in the cell.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) a method of treating a subject for beta cell destruction comprising contacting said subject with an agent that reduces levels of ceramide in the cells of said subject as compared to the untreated ceramide level, wherein reduction in ceramide levels protects cells of th subject against ceramide-induced lipotoxicity;

(2) a method for preventing non-insulin dependent diabetes mellitus (NIDDM) in a subject, comprises:

(a) identifying a subject at risk of diabetes mellitus; and

(b) providing to the subject a composition comprising an agent that reduces levels of ceramide in the cells of said subject as compared to the untreated ceramide level, wherein the reduction in ceramide level

protects

cells against ceramide-mediated apoptosis of beta -cells;

(3) a method for ***inhibiting*** ceramide-mediated lipotoxicity of a target cell comprising blocking ceramide production or

accumulation

in the cell; and

(4) a method of preventing non-insulin dependent diabetes mellitus (NIDDM) in a subject comprising blocking ceramide production in a pancreatic beta cell in the subject, wherein blocking ceramide

production

is accomplished by administering to the cell an amount of an inhibitory agent sufficient to protect the cell from ceramide-mediated lipotoxicity.

USE - The process is used to treat a pathologic condition selected from insulin-dependent diabetes mellitus (IDDM)insulin-independent diabetes mellitus (NIDDM) and obesity (all claimed).

ADVANTAGE - The composition is useful for alleviating the deleterious

effects of beta -cell dysfunction and beta -cell destruction caused by lipotoxicity.

Dwg.0/6

L11 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1996:113481 CAPLUS

DOCUMENT NUMBER: 124:137837

TITLE: Host cells transformed with fusion protein gene and method for screening test samples with receptor-ligand interactions or peptide-binding activities

INVENTOR(S): Young, Kathleen H.; Ozenberger, Bradley A.

PATENT ASSIGNEE(S): American Cyanamid Co., USA

SOURCE: PCT Int. Appl., 54 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9534646 A1		19951221	WO 1995-US6895	19950531
W: AM, AU, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IS,				

JP, KG, KP, KR, KZ, LK, LR, LT, LV, MD, MG, MN, MX, NO, NZ, PL, RO, RU,

SG, SI, SK, TJ, TM, TT, UA, UG, UZ, VN

RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT,

LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE,

SN, TD, TG

US 5989808 A 19991123 US 1994-259609 19940614 CA 2195083 AA 19951221 CA 1995-2195083 19950531

AU 9526066 A1 19960105 AU 1995-26066 19950531 AU 706173 B2 19990610

EP 765389 A1 19970402 EP 1995-920689 19950531 EP 765389 B1 20030716

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE

RU 2208646 C2 20030720 RU 1997-100776 19950531 AT 245189 E 20030815 AT 1995-920689 19950531

ZA 9504892 A 19960130 ZA 1995-4892 19950613 LT 4230 B 19971027 LT 1997-4 19970113

LV 11906 B 19980620 LV 1997-4 19970214 US 6251602 B1 20010626 US 1999-263944 19990308

US 6284519 B1 20010904 US 1999-305483 19990506 PRIORITY APPLN. INFO.: US 1994-259609 A 19940614

WO 1995-US6895 U 19950531

AB This invention relates to novel modified host cells which express heterologous fused proteins and methods of screening for test samples

having peptide-binding activity; wherein the modified host cell comprises:

(a) a gene sequence encoding a heterologous fusion protein; said fusion protein comprising a first peptide of a peptide binding pair, or segment of said first peptide, which is joined to either a DNA binding domain or its corresponding transcriptional activation domain of a transcriptional

activation protein; (b) a gene sequence encoding a heterologous fusion protein, said fusion protein comprising a second peptide of the peptide binding pair in (a), or a segment thereof, fused to either a DNA binding domain or its corresponding transcriptional activation domain,

whichever

one is not employed in (a); (c) a reporter gene operatively assocd. with the transcriptional activation protein, or a portion thereof; (d) optionally, a deletion or mutation in the chromosomal DNA of the host cell

for the transcriptional activation protein if present in the selected host cell.

L11 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1993:227525 CAPLUS

DOCUMENT NUMBER: 118:227525

TITLE: Ecdysteroid dependent regulation of genes in

mammalian

cells

INVENTOR(S): Godowski, Paul J.

SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9303162	A1	19930218	WO 1992-US6391	19920803
W: JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, SE				
EP 598011	A1	19940525	EP 1992-917507	19920803
EP 598011	B1	19981111		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, SE				
JP 07501928	T2	19950302	JP 1992-503762	19920803
AT 173295	E	19981115	AT 1992-917507	19920803
ES 2123569	T3	19990116	ES 1992-917507	19920803
PRIORITY APPLN. INFO.: US 1991-742127 19910808				
WO 1992-US6391 19920803				

AB A method of inducing gene expression in a mammalian cell comprises contacting an ecdysteroid receptor with an ecdysteroid within a mammalian cell contg. a gene linked to an ecdysteroid response element. The *Drosophila* ecdysteroid receptor cDNA was cloned and sequenced. Human 293 cells were cotransfected with a vector contg. this cDNA and a vector in which the chloramphenicol acetyltransferase (CAT) gene was linked to 4 ecdysone response elements. Cat activity was stimulated in cell exts. incubated with muristerone A, but not with .alpha.- or 20-hydroxy-ecdysone or polypodine B, nor with dexamethasone, thyroid hormone, retinoic acid, etc.

L11 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1993:226597 CAPLUS
DOCUMENT NUMBER: 118:226597
TITLE: Selecting agonists and antagonists for growth hormone-like ligands which form ternary complexes with receptors

INVENTOR(S): Cunningham, Brian C.; Devos, Abraham M.; Mulkern,

Michael G.; Ultsch, Mark; Wells, James

PATENT ASSIGNEE(S): Genentech, Inc., USA

SOURCE: PCT Int. Appl., 77 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9221029	A1	19921126	WO 1992-US3743	19920506
W: JP, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
EP 586549	A1	19940316	EP 1992-912746	19920506
EP 586549	B1	20000920		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
JP 06507715	T2	19940901	JP 1993-500070	19920506
JP 3417558	B2	20030616		
AT 196548	E	20001015	AT 1992-912746	19920506
JP 2001270837	A2	20011002	JP 2001-54464	19920506
JP 2003159061	A2	20030603	JP 2002-271918	19920506
US 5506107	A	19960409	US 1993-122548	19930929
US 6429186	B1	20020806	US 1994-308879	19940919
PRIORITY APPLN. INFO.: US 1991-698753 A 19910510				
US 1992-864120 A2 19920406				
JP 1993-500070 A3 19920506				
WO 1992-US3743 W 19920506				
US 1993-20327 B1 19930219				

AB Growth hormones (GHs), and the class of conformational ligands to which they belong [including erythropoietin, placental ***lactogen***, prolactin, .alpha.- and .beta.- ***interferons***, colony-stimulating factors (CSFs), and interleukins 2, 3, 4, 6, and 7], bind sequentially through 2 different receptor-binding sites to 2 receptor mols. to form a 1:2 complex. Formation of such a complex is useful as a criterion for whether a candidate compd. is a ligand agonist or antagonist. The candidate compd. may be a (recombinant) ligand mol. with an altered amino acid sequence whose affinity for the receptor at one or both binding sites is altered. A monoclonal antibody to glycosylated GH receptor was identified which induced body wt. gain in hypophysectomized rats. A cell proliferation assay for GH agonists was developed which used granulocyte-CSF (G-CSF)-responsive FDC-P1 cells transfected with a vector contg. hybrid cDNA for the GH-binding domain of the GH receptor and part of the G-CSF receptor lacking the G-CSF-binding domain.